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Plasma Volume Shifts and Exercise Thermoregulation with Water Immersion and Six-Degree Head-Down Tilt

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Nomenclature

[Hb]	hemoglobin concentration
Hct	hematocrit
$[K^+]$	potassium concentration
$[Na^+]$	sodium concentration
rh	relative humidity
T_{es}	esophageal temperature, degrees Centigrade (°C)
T_{re}	rectal temperature, °C
$\overline{T}_{sk}$	mean skin temperature, °C
$\dot{V}O_2$	oxygen consumption
$\dot{V}O_{2max}$	maximum oxygen consumption
$\dot{V}O_{2peak}$	peak oxygen consumption

Acronyms

ANOVA	analysis of variance
BV	blood volume
ECF	extracellular fluid
FVR	forearm vascular resistance
HDT	head-down tilt
HDT1	1-hour (hr) 6-degree head-down tilt
HDT24	24-hr 6-degree head-down tilt
ICF	intracellular fluid
LBNP	lower body negative pressure
NS	nonsignificant
PV	plasma volume
SD	standard deviation
SE	standard error
SkBF	skin blood flow
SBV	skin blood velocity
WI	water immersion

Plasma Volume Shifts and Exercise Thermoregulation with Water Immersion and Six-Degree Head-Down Tilt

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Summary

The hypothesized fluid shifts and resultant responses that occur during spaceflight are simulated by six-degree head-down tilt (HDT) and water immersion (WI). An initial response is a transient increase in plasma volume (PV). Blood volume regulating mechanisms subsequently reduce the PV; with HDT the greatest reduction occurs in 24 hours (hr), but with WI the effect is rapid and occurs within several hr. Seven male subjects were studied in two experiments: 1) Exercise thermoregulation in conditions of increased PV (1-hr HDT (HDT1)) and reduced PV (24-hr HDT (HDT24)); and 2) where WI was used to reproduce the reduced PV of 24-hr HDT.

It is hypothesized that exercise rectal temperature (T_{re}) would be higher following HDT24 than following HDT1. HDT began after 60 minutes (min) of chair rest. The PV increased by 6.1% with HDT1 and decreased by 4.3% with HDT24. After these treatments the subjects performed 70 min of supine cycling at 58% of peak oxygen consumption ($\dot{V}O_{2peak}$) at 22.0 degrees Centigrade (°C) and 47% relative humidity (rh). Preexercise T_{re} was higher after HDT24, and this relative difference was not altered with exercise. Initial mean skin temperatures ($\overline{T}_{sk}$) were higher in HDT24 but leveled off after 30 min of exercise, inferring a reduced skin heat conductance. The $\overline{T}_{sk}$ continued to increase throughout the exercise in HDT1. An elevated preexercise T_{re} causes thresholds for thermoregulatory increases in skin blood flow (SkBF) to be reached earlier and also causes earlier sweating. In contrast, reductions in PV increase the threshold values for thermoregulatory responses. These interactions apparently caused parallel increases in T_{re} during exercise in the present experiment, suggesting no alteration in the overall thermoregulatory response at a given T_{re}.

In the second experiment, WI began at 0900 hr to synchronize the end of WI with HDT24. An early morning hemodilution was evidenced by lower hematocrit (Hct) and hemoglobin concentration ([Hb]) during preimmersion. Changes in PV were calculated relative to this baseline. After 3 to 6 hr of WI, PV decreased by 1.1% as compared with a decrease of 4.3% with HDT24. Individual PV responses varied from −14.9% to +8.9% between WI and HDT24. Time of day and intraindividual responses to WI and HDT24 greatly affected PV changes. Evaluation of the similarity of these two models indicates that there are differences in the overall PV response and reproducibility.

1.0 Introduction

During spaceflight, astronauts are no longer exposed to the effects of Earth's gravity. There is a headward fluid shift estimated at approximately 2 liters (Levy and Talbot, 1983). A negative fluid balance is hypothesized to result in response to this fluid redistribution, and it is reflected in a reduced plasma volume (PV). The reduced PV is hypothesized to result from a modest diuresis, natriuresis, and reduced fluid intake during flight (Greenleaf, 1986). These losses are associated with reduced exercise capacity and orthostatic intolerance after flight (Levy and Talbot, 1983).

Experimental models used to simulate the microgravity environment of space include head-down tilt (HDT) and water immersion (WI). These weightlessness, but Earth-bound, analogs also induce a headward shift of body fluids from the lower body to central reservoirs when compared to a normal upright posture under Earth's gravitational influence (Blomqvist and Stone, 1983). A seated upright posture has been suggested as the only valid pre-HDT control position since a supine posture already has the influence of the fluid-shifted state affecting the system (Gharib et al., 1988). Similar to HDT, the effects of WI are more pronounced when an upright control is used (Harrison et al., 1986). Physiologic adjustments to the headward shift of fluid include a transient increase in PV (hypervolemia) and an ensuing volume regulating diuresis and natriuresis (Greenleaf et al., 1981; Gharib et al., 1988). The result of this compensatory response is that PV, and therefore total intravascular volume, is maintained at a lower level (hypovolemia). Various endocrine systems have been hypothesized to mediate this exchange and physiological balance of fluid and sodium. Suppression of arginine vasopressin release and the

renin-angiotensin-aldosterone system and stimulation of atrial natriuretic factor release have been proposed as possible mediators (Levy and Talbot, 1983; Blomqvist and Stone, 1983; Greenleaf, 1986). The resultant decrease in PV accounts for approximately one-third of the early weight loss associated with spaceflight, bed rest, and WI (Blomqvist and Stone, 1983). Nixon et al. (1979) have shown a 0.4-liter decrease in blood volume (BV) following 6 hours (hr) of 5-degree (deg) HDT by using a supine control period as reference, with a 0.5-liter decrease after 24 hr. Changes in body weight during HDT bed rest and computer modeling suggest that the decrease in body fluids and PV with HDT occurs within 24 hr (Greenleaf et al., 1989; Simanonok et al., 1993). Because the hemodynamic adaptation to simulated microgravity by HDT is essentially complete by 24 hr, HDT is an attractive model for use in studies of BV regulation. With WI, the PV follows a similar, but more rapid, time course of transient hypervolemia followed by a hypovolemia consequent to the diuresis and natriuresis (McCally, 1964; Greenleaf et al., 1981).

The decrease in PV, an aspect of the cardiovascular deconditioning following HDT bed rest or WI, has been implicated in the impaired thermoregulatory response to exercise (Greenleaf and Reese, 1980; Greenleaf et al., 1985). In contrast, an increase in PV has been reported consequent to appropriate exercise stimuli in ambulatory subjects, and it results in enhanced thermoregulatory and cardiovascular stability (Convertino, 1991). Increases in PV result in maintaining core temperature at a lower level, while heart rate is reduced and stroke volume is increased at a given exercise intensity (Fortney et al., 1983; Green et al., 1990).

The idea for the present study originated following work on the 1986 NASA Ames Research Center Exercise Bed-Rest Study in which it was determined that the normal loss of PV during 30 days of 6-deg HDT bed rest could be completely attenuated with an appropriate exercise stimulus. Subjects who utilized variable-intensity supine ergometry (60%–90% of peak oxygen consumption ($\dot{V}O_{2peak}$)) for 30 minutes (min) twice daily were able to maintain aerobic work capacity (+2.6%) and PV (–1.5%), not significantly different from ambulatory levels. This maintenance is in contrast to control subjects who experienced decreases of 18.2% and 16.8% for $\dot{V}O_{2peak}$ and PV, respectively (Greenleaf et al., 1989, 1992).

This evidence that PV can be maintained at ambulatory levels during HDT bed rest with exercise suggests that PV is an aspect of the spaceflight deconditioning that can be attenuated with an appropriate exercise prescription. The bed rest period selected in the present study was 24 hr, long enough to cause significant changes in the PV yet minimizing the manifestations of other effects of HDT deconditioning.

A second issue of interest addressed in this study is exercise hemoconcentration. Although it has been hypothesized that increases in mean arterial pressure and the number of perfused capillaries in working muscle would result in a net efflux of plasma from the vascular space, observations have varied considerably (Harrison, 1985). There appears to be an upper limit of the magnitude of plasma efflux resulting from exercise such that the acute effect is dependent on preexercise conditions; e.g., the reference posture and the ambient temperature (Diaz et al., 1979; Hagan et al., 1980). An upright posture and/or heat exposure induces hemoconcentration, which can attenuate any further hemoconcentration during exercise. The PV, governed by the balance of Starling forces across the capillaries and postcapillary venules (Harrison, 1985), is defended at a minimum level under these conditions. As previously noted, the central BV expansion and the transient hemodilution that occurs with WI or HDT is compensated by a volume correcting diuresis and natriuresis. Whether or not the PV shift of exercise would be attenuated after PV is reduced consequent to an HDT-induced diuresis is unknown. In the present study the relative change in PV in response to exercise will be compared following hypervolemia and hypovolemia induced by a chronic adjustment to the same HDT posture.

Both HDT and WI attenuate gravitational hydrostatic gradients in the body and cause a headward shift of body fluids. A significant difference is that the cardiovascular effects of WI are more immediate and powerful than those of 6-deg HDT (Blomqvist and Stone, 1983).

Statement of the Problem

The experimental design used in this study was to test subjects in two related, but independent, phases. In phase I, exercise thermoregulation was studied in subjects with hypervolemic and hypovolemic changes in PV induced by 1-hr (HDT1) and 24-hr HDT (HDT24), respectively. With a seated reference posture, HDT1 was used to induce an acute postural hypervolemia via a normal redistribution of the extracellular fluid volume. The hypovolemic condition in phase I was produced by HDT24, which allowed adequate time for a diuresis and natriuresis to occur as compared with HDT1. Thermoregulatory responses to exercise were tested during submaximal exercise under these two conditions. Conclusions based on the results of the phase I portion of the study are dependent on two assumptions: 1) the primary change that affects exercise thermoregulation following HDT24 is the decrease in PV; and 2) changes in PV during

HDT can be estimated accurately from calculated chair-rest control Hct and [Hb]. In phase II, an attempt was made to reproduce the hypovolemia of HDT24 by using WI. Since HDT and WI are both used to simulate exposure to weightlessness, the common salient feature of changes in PV needs to be characterized in the two models. An assumption made with respect to phase II was that the level of hypovolemia caused by HDT24 could be reliably reproduced with less than 6 hr of WI.

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2.0 Review of Literature

Autonomic Mediated Thermal Adjustments to Changes in Posture and Vascular Volume

The body compensates for changes in posture by inducing cardiovascular responses through both central and local reflexes (Rowell, 1986). Central factors include the high pressure arterial and low pressure cardiopulmonary baroreceptors. Reflex alterations in autonomic nervous system outflow provide control of arteriolar resistance, venous tone, heart rate, and contractility. In addition, neuroendocrine mediated changes occur in plasma renin and angiotensin II levels (Rowell, 1986). Local adjustments are associated with venous-arteriolar reflexes and the autoregulatory function of the arteriolar smooth muscle (Blomqvist and Stone, 1983).

Asmussen et al. (1940) observed changes in body temperatures when a subject assumed a supine posture from an upright reference. After 1 hr in a supine posture, the rectal temperature (T_{re}) was 0.5°C lower and the mean skin temperature ($\overline{T}_{sk}$) was 1.0°C higher, both reflective of changes in heat conductance related to skin blood flow (SkBF). Amberson (1943) used this and other evidence to conclude that cutaneous vasoconstriction and a diminished rate of heat loss occurs in the upright posture. He cited the possibility that the signals for orthostatic vasoconstriction might originate in the brain.

Recent evidence confirms the presence of a cutaneous vasoconstriction that is mediated through the autonomic nervous system. Mano (1990), in a review of microneurographic studies, reported that sympathetic nerve activity is increased from supine to sitting to standing and from supine to upright with head-up tilting. He suggested that the unloading of arterial and cardiopulmonary receptors was responsible for increased sympathetic activity with the gravitational stress of an upright posture. The increased sympathetic activity associated with an upright posture accelerates the heart rate, increases cardiac output, and increases peripheral vascular resistance.

Pannier et al. (1991), induced a headward shift of body fluids with 5-deg HDT; a diuresis and natriuresis followed, resulting in a reduced PV. Forearm vascular resistance and venous tone, measured with venous occlusion plethysmography, decreased for up to 6 hr after the assumption of HDT, but by the end of 24 hr, the parameters had returned to pretilt values. The authors stated that the return of these variables toward the baseline was expected to maintain cardiovascular homeostasis with the decrease in total intravascular volume. The associated changes in PV were not reported, but results suggest that the degree of PV reduction with HDT24 can cause alterations in autonomic outflow. An initial decrease in sympathetic outflow and decreased vascular resistance would be associated with the transient hypervolemia induced by HDT.

Thompson et al. (1990) investigated baroreflex responses to changes in BV. An isosmotic hypovolemia of 16% resulted from administration of diuretics, whereas a 0.9% saline solution increased PV by 9.5%. Analysis of the stimulus-response curve of the carotid-cardiac baroreflex demonstrated no significant differences of normovolemia from hypervolemia or hypovolemia. The conclusion was that high-pressure arterial baroreflexes (carotid) were not affected by hypovolemia under conditions of varied volemic states. Cardiopulmonary reflexes, on the other hand, were sensitive to changes in PV. Cardiopulmonary reflexes were evaluated by measuring forearm vascular resistance (FVR) with venous occlusion plethysmography during progressive increases in lower body negative pressure (LBNP). Hypovolemia resulted in an increased resting FVR and an upward shifting of the response to LBNP. Thus, the threshold for initiation of cardiopulmonary reflexes in response to LBNP was reached at a lower level of stress. This observation was supported by other findings from the same laboratory: reductions in PV resulted in a greater baseline vasoconstriction. The sensitivity of the reflexes is not necessarily altered, but the threshold for response is reached earlier because of the reduced PV and cardiac filling pressure.

Body Temperature Responses to Exercise

Metabolic heat production increases during exercise, creating an endogenous heat load, which then increases body temperature. Common sites at which deep body temperature is measured give different response times and, to an extent, equilibrium values. Esophageal temperature (T_{es}) and the T_{re} are used to determine core-temperature responses to exercise. Esophageal temperature responds faster than T_{re} and stabilizes in 15 to 25 min of exercise (Nielsen and Nielsen, 1962). The T_{es} also stabilizes at a lower temperature during leg exercise, and it is probably a good estimation of aortic blood and heart temperatures (Saltin and Hermansen, 1966; Nielsen and Nielsen, 1962). The T_{re} responds more slowly than the T_{es}, probably because of the large heat capacity relative to blood flow in the tissues of the rectum (Nielsen and Nielsen, 1962). The temperature gradient in the rectum shows no substantial differences measured at depths of 12 to 27 cm (Nielsen and Nielsen, 1962). The finding that T_{re} is higher most likely reflects heating caused by blood returning from working leg muscles (Nielsen and Nielsen, 1962). It is evident that if T_{re} is used as an index of changes in core temperature during exercise the

duration of exercise must be longer and direct comparisons cannot be made to T_{es}.

Marius Nielsen (1970) summarized a series of studies he and co-workers performed to determine core-temperature responses to exercise and ambient conditions. Body temperature is regulated at a higher temperature during exercise than at rest. When core temperature is plotted as a function of dynamic exercise at increasing power output expressed as $\dot{V}O_2$, a positive linear relationship is seen. Subjects with higher maximum power output exhibit a lower temperature response at any given absolute $\dot{V}O_2$. When this relationship is expressed as the relative percentage of maximum $\dot{V}O_2$ as a function of core temperature, the difference between subjects is minimized (Saltin and Hermansen, 1966).

Thermoregulatory Responses to Exercise

Exercise performed at environmental temperatures from 5°C to 30°C (with ambient air flow) demonstrates the relative independence of core temperature with respect to environmental temperature (Nielsen, 1970). For this independence to occur, thermoregulatory mechanisms must be utilized to increase heat dissipation. Thermoregulatory responses to endogenous and ambient heat loads include a shunting of circulating blood to the cutaneous circulation for conductive heat exchange and sweating to provide for evaporative heat loss (Gisolfi and Wenger, 1984; Rowell, 1986). Attenuation of either mechanism can result in higher core temperatures during exercise.

Body temperature rises before mechanisms are invoked to regulate the temperature at a new level. Since the body temperature during exercise rises to a new relatively stable level independent of ambient heat stress between 5°C and 30°C, one hypothesis suggests a change in the set-point regulation of body temperature. Gisolfi and Wenger (1984) refer to a "set point" as a conceptual model of a central integrator receiving information from skin and core thermal receptors and generating a central command signal to control thermoregulatory effector responses. When a heat stress is applied and body temperatures (core and skin) rise, effector mechanisms are recruited as these temperature thresholds are reached. This closed-loop, integrated, negative-feedback system maintains core temperature at a higher level. The increased core temperature serves several possible functions in the body. With a greater increase in core temperature than in skin temperature, the gradient for conductive heat transfer from the core to the skin is greater. The increase in skin temperature also aids in evaporation of sweat from the skin. With an increase in ambient temperature, the decreases in heat loss by radiation and convection are balanced by the increase in evaporative heat loss (Nielsen, 1970). Skin blood flow is controlled by both thermoregulatory inputs from core and skin receptors and nonthermoregulatory mechanisms such as baroreflexes. These inputs allow for a superimposed effect of central cardiovascular control on thermoregulatory mechanisms (Johnson, 1986; Rowell, 1986).

Sawka (1992) and Nielsen (1984) concluded that hypovolemia results in decreases in SkBF, stroke volume, cardiac output, and increases in heart rate; increases in plasma osmolality (P_{osm}), on the other hand, often decrease sweat rate. Although these effects on exercise thermoregulation are exacerbated in the heat, they are also evident in temperate conditions (Nielsen, 1984). In the following sections some literature related to these interpretations and observations is reviewed.

Hypovolemic Effects on Skin Blood Flow and Sweating

Hypovolemia induced by diuretics leads to an isosmotic reduction in PV. Diuretics promote increased urine excretion primarily by a decrease in the reabsorption of sodium and by the osmotic effect of sodium on water. An isosmotic decrease in body fluids does not produce a solute excess in the extracellular fluid (ECF), which would in turn cause a shift of fluid from the intracellular compartment (ICF). This isosmotic decrease leads to a greater loss of fluid from the ECF and PV than when hypovolemia from exercise or heat exposure is induced by hypotonic sweat loss (Sawka, 1992). Increased plasma osmolality ensues. Water then moves from the intracellular space, and cells become dehydrated. Hypotonic fluid loss can elevate core temperature and reduce sweating responses independent of body-water gain or loss (Sawka, 1992). Since no osmotic changes occur in the body following diuretic use, the effects of reduction in PV can be separated from changes in plasma osmolality. Results from four studies illustrate the effects of an isosmotic reduction of PV by diuretics or bed rest on exercise thermoregulation.

Claremont et al. (1976) studied subjects in hydrated and hypovolemic conditions. A rapid 15.3% isosmotic reduction in PV was achieved by an oral administration of 60 to 80 milligrams (mg) of Lasix. Seven subjects performed 2 hr of exercise at 39% $\dot{V}O_{2\,max}$ at 39°C and 35% relative humidity (rh). Rectal temperature and muscle temperatures were significantly higher in the hypovolemic group as compared to the control group. The $\overline{T}_{sk}$ and calculated skin conductance were lower in the hypovolemic group. Exercise sweat rates were similar in both groups because of the lack of change in plasma osmolality prior to exercise. The researchers concluded that the

elevated core temperatures in the hypovolemic group were caused by reductions in skin and muscle blood flow, which reduced the conductance of heat from core to skin.

Nadel et al. (1980) reduced PV isosmotically by 17.5% with diuretics; cycling exercise was performed at 55% of $\dot{V}O_{2\,max}$ at 35°C. Compared with hydrated control, the threshold core temperature for cutaneous vasodilation was higher and onset of increased SkBF was delayed, resulting in reduced heat transfer from core to skin and a higher core temperature. However, the slope of the increase in SkBF as compared with T_{es} was unchanged with hypovolemia. After 30 min the heart rate was 6 beats per minute (bpm) higher, the stroke volume was 17 milliliters (ml) lower, and the cardiac output was lower in the hypovolemia group as compared to the control group. The reduced SkBF in hypovolemia was attributed to an increase in baroreflex mediated cutaneous vasoconstrictor activity that predominated the vasodilator drive due to thermal effects. Maximal levels of SkBF were also reduced during hypovolemic exercise. This vasoconstriction served to maintain cardiac filling pressure in the face of a reduced circulating BV and, as a consequence, reduced conductive heat transfer and loss. As stated earlier, once the threshold for vasodilation was reached, the increase in SkBF per unit increase in T_{es} for the hypovolemic group was similar to that for the control group (i.e., no change in slope). Since the threshold and not the sensitivity of the response was affected and other thermoregulatory inputs were assumed to be similar in the two groups, the response was attributed to a central nervous system effect. Although sweating responses were not reported, if the change were in the central nervous system, sweating threshold would be expected to be similarly affected by baroreflex activity.

Fortney et al. (1981) reduced PV isosmotically by 14.9% with diuretics before subjects exercised at 65% to 70% of $\dot{V}O_{2\,max}$ at 30°C, 40% rh. The slope of the sweat rate as a function of T_{es} was reduced over inactive muscles with hypovolemia while the threshold for sweat onset was unchanged. This effect was independent of changes in plasma osmolality, which generally affects sweat rate. One interpretation of the hypovolemia-induced reduction in sweat rate is in agreement with findings from the previous study, in which nonthermoregulatory inputs affected the central effector drive to thermoregulatory mechanisms. The exercise intensity of Fortney's study of 65% to 70% was higher than the 39% $\dot{V}O_{2\,max}$ used by Claremont et al. (1976), in which sweat rate was unaffected by hypovolemia. The total thermoregulatory stress caused by hypovolemia, ambient heat, and exercise intensity may be necessary to affect sweat rate.

In a study of exercise thermoregulation following 14 days of bed rest a hypovolemia of 15.1% occurred in the no-exercise control group (Greenleaf and Reese, 1980). Compared to ambulatory control, exercise resulted in higher equilibrium T_{re} and lower $\overline{T}_{sk}$ and calculated skin heat conductance. The authors suggested that the variable heat transfer from core to periphery is a thermoregulatory mechanism reflected in the changes in skin conductances and $\overline{T}_{sk}$. The same absolute load was utilized for exercise before and after bed rest. After 14 days of bed-rest deconditioning, the relative exercise intensity was 45.4% to 48.4% of $\dot{V}O_{2\,max}$, compared to 43.3% in the ambulatory control group. Thermoregulatory responses are dependent on the relative intensity of exercise, and T_{re} is expected to increase 0.03°C for each percentage increase in relative intensity (Saltin and Hermansen, 1966). When the authors compared the differences in exercise T_{re} and ambulatory T_{re}, the increase in relative exercise intensity would have to have been several times as great (14% for an isometric exercise group) as the actual relative 2% to 5% difference in percent of $\dot{V}O_{2\,max}$.

An isosmotic reduction in PV prior to exercise effects a change in thermoregulatory mechanisms that is manifest by reductions in SkBF. In severe exercise and heat stress, sweating can also be reduced. Both of these changes in thermoregulatory behavior serve to maintain cardiac filling and blood pressures despite compromising thermoregulatory mechanisms, resulting in greater increases in core temperature (Rowell, 1986).

Plasma Volume Changes with Exercise

Acute plasma volume responses to exercise– Local vasodilation in working muscles during exercise along with increases in mean arterial pressure and osmotic changes in active muscle can lead to a PV efflux and net hemoconcentration (Senay and Pivarnik, 1985). Hydrostatic gradients caused by gravity play a critical role in determining the net change. Hagan et al. (1980) tested subjects in a seated-upright and a low-sit posture (seated with legs horizontal) during three 60-min exercise bouts at 31%, 51%, and 69% $\dot{V}O_{2\,max}$. Prior to exercise (relative to supine) postural hemoconcentrations of 6.9% and 14.1% resulted from low-sit and seated-upright postures, respectively. Exercise in the low-sit posture caused an additional decrease in PV relative to intensity, but in the seated-upright posture exercise had minimal further effect. The authors concluded that acute PV changes with exercise are greatly influenced by the reference posture and the time spent stabilizing in that posture. The addition of hydrostatic gradients with a seated-upright posture

causes a postural hemoconcentration that imposes a constraint on any further changes with exercise.

Exercise-induced hypervolemia– The transient contraction and expansion of PV as a result of exercise and thermal stimuli can lead to an increased total vascular volume. These increases in BV and PV lead to increased thermoregulatory and cardiovascular stability during exercise (Convertino, 1991). Graded cycling exercise above 40% of maximum oxygen consumption significantly increases plasma arginine vasopressin concentration and plasma renin activity (which plays a regulatory role in aldosterone secretion (Convertino et al., 1981)). When these systems are stimulated, total body water increases via the action of 1) arginine vasopressin through its effect on renal water reabsorption; 2) aldosterone through sodium reabsorption directly; and 3) water reabsorption indirectly. Plasma proteins and sodium are major osmotic factors in the maintenance of PV. Total plasma albumin increases with exercise training in conjunction with increases in PV (Convertino et al., 1980). Chronic stimulation of these systems by exercise may play a role in the long-term regulation of BV (Convertino et al., 1980). The decrease in PV with HDT bed rest can be reversed with an exercise-induced hypervolemia. Subjects performing short duration (30-min sessions twice daily), high-intensity exercise of up to 90% of peak $\dot{V}O_2$ maintained PV at ambulatory levels after 30 days of HDT bed rest (Greenleaf et al., 1989). In contrast, subjects in both the control (no exercise) and isokinetic maximal exercise groups had significantly reduced PV throughout bed rest. Exercise training that provides sufficient thermal and exercise factors produces an increase in PV which, during bed rest, can override decreases in PV normally seen.

An hypothesis on the influence of hydrostatic-induced hypervolemia on exercise thermoregulation– Nielsen et al. (1984), utilizing WI to induce a headward shift of fluid, have hypothesized an enhanced ability in this condition to respond to the cardiovascular stress of exercise. Subjects were immersed in an upright posture to the level of the xiphoid process. The hydrostatic counterpressure provided by WI prevented blood redistribution to dependent regions. Conditions of seated and upright cycling exercise at 50% of maximal $\dot{V}O_2$ were compared when subjects were in either 35°C water or 45°C air ($\overline{T}_{sk}$ of 35°C). The prevention of dependent venous pooling and the resulting central BV expansion allowed cardiac output to be maintained during submaximal exercise and provided a greater potential to shunt blood to the cutaneous circulation. Forearm blood flow was higher during WI exercise and continued to increase throughout exercise; whereas in air, blood flow plateaued at a lower level. Thus, the authors hypothesized that the thermoregulatory effect on SkBF and cardiac output, which normally enhances exercise capacity, will be present in microgravity environments until the system is adjusted by a loss of PV.

Conclusion

Changes in posture can affect thermoregulation at rest, affecting both skin and core temperatures. Heat loss will increase when gravitationally induced hydrostatic gradients (e.g., a supine posture) are reduced. The acute increases in central BV and PV decrease sympathetic nervous system activity and mediate increases in SkBF. With consequent increased conductive heat loss, core temperature decreases. Isosmotic reductions in PV have superimposed effects on postural changes in thermoregulation. With a reduced PV the cardiopulmonary reflexes increase sympathetic outflow and thus increase threshold values for thermoregulatory increases in SkBF and sweating. These variations in thermoregulatory behavior are often evident during the increased endogenous heat stress of exercise. Exercise training can increase PV chronically and it will provide greater body fluid for sweating and a larger vascular volume for subsequent redistribution to the cutaneous circulation. It is hypothesized that, during conditions of acute postural hypervolemia, thermoregulatory behavior will be enhanced. Maintenance of this hypervolemia during spaceflight may be an important consideration in exercise prescriptions.

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3.0 Exercise Thermoregulation After HDT1 and HDT24 in Men

Summary

Seven men (31 ± 6 year (yr) standard deviation (SD)) were studied during 70 min of supine cycling at 58% peak $\dot{V}O_2$ (1.99 ± 0.10 l· min^{-1}) at 22.0°C and 47% rh. The hypothesis was that HDT24 and the associated decrease in PV would result in higher exercise T_{re} compared to a hypervolemic condition of HDT1. Relative to pretilt chair rest, HDT1 resulted in a 6.1% increase and HDT24 in a 4.3% decrease in PV (probability (p) < 0.04). The plasma osmolality remained unchanged in HDT1 and HDT24 (nonsignificant (NS) p = 1.00). The preexercise T_{re} for HDT1 and HDT24 were significantly different (p < 0.04): 36.71°C ± 0.06 for HDT1 and 36.93°C ± 0.11 for HDT24. Seventy min of exercise did not alter this relationship (NS, p < 0.11), with increases in T_{re} of 1.30°C (HDT1) and 1.33°C (HDT24). A significant time-treatment interaction (p < 0.003) occurred in $\overline{T}_{sk}$ during exercise. After min 30 in HDT24, $\overline{T}_{sk}$ leveled off at 31.1°C, whereas in HDT1 it continued to rise, reaching 31.5°C at min 70. Changes in body weight during exercise were not significantly different (NS, p < 0.14): –1.63 ± 0.24 kilogram (kg) for HDT1 and –1.33 ± 0.09 kg for HDT24. The effects of an elevated preexercise T_{re} and reduced PV in HDT24 may have had offsetting effects on thermoregulatory mechanisms leading to no difference in the increases in T_{re} during exercise.

Introduction

One method of simulating the hypothesized fluid shifts and resultant responses that occur during spaceflight is the change in posture of 6-deg HDT (Blomqvist and Stone, 1983). With HDT a transient increase in PV is followed by a natriuresis and diuresis resulting in a decreased PV. Both body weight changes during HDT bed rest and results from computer modeling suggest that the decrease in PV with HDT occurs within 24 to 48 hr (Greenleaf et al., 1992; Simanonok et al., 1993). If changes in PV induced by microgravity analogs negatively affect thermoregulatory behavior during exercise, exercise prescriptions for astronauts might include emphasis on the maintenance of PV.

The ability to regulate core temperature during exercise is dependent on evaporative heat loss through sweating and conductive heat transfer from the core to the skin to the ambient environment. Increases in SkBF and the onset and rate of sweating are the controlled determinants of heat loss during exercise. Thermal inputs from the skin and core influence changes in SkBF and the onset and rate of sweating (Gisolfi and Wenger, 1984; Rowell, 1986). In addition, SkBF is dependent on the sympathetic outflow influenced by posture and the level of the BV. Increases in sympathetic activity and subsequent cutaneous vasoconstriction can be caused by an upright posture or hypovolemia (Johnson, 1986).

An isosmotic reduction of PV, such as that caused by diuretics, negatively affects circulatory and SkBF during exercise, but it does not affect the relationship of core temperature to the onset of sweating (Fortney et al., 1981). Decreases in PV caused by exercise, heat, or limiting fluid intake result in both a reduction in PV and an increase in plasma osmolality, which inhibits sweating during subsequent exercise and results in higher core temperatures (Greenleaf and Castle, 1971; Fortney et al., 1984).

In the present study HDT was used to simulate reduced hydrostatic effects of gravity during spaceflight. The primary adjustments that occur within 24 hr of HDT are assumed to be vascular fluid redistribution and the subsequent reduction in PV. A compromised ability to thermoregulate during exercise would support the importance of maintaining PV during spaceflight.

Methods

Subjects– Seven male subjects participated in the study; their mean ± SD values are: age, 31 ± 6 yr, height, 182 ± 5 centimeters (cm), weight, 78.6 ± 9.0 kg; and peak $\dot{V}O_2$, 3.42 ± 0.34 l · min^{-1} (44 ± 6 ml · kg^{-1} · min^{-1}). All subjects signed an informed consent statement approved by NASA Ames Research Center's Human Research Experiments Review Board and the Human Subjects Committee, University of California, Davis. Each subject passed a comprehensive medical examination and was thoroughly familiarized with the procedures to be used. In order to characterize subjects and determine power output for experimental sessions, testing for peak $\dot{V}O_2$ was performed at least twice. All exercise was performed in the supine posture on a Quinton model 846T Imaging/Ergometer Table (Quinton Co., Seattle, WA 98121). The protocol for peak $\dot{V}O_2$ utilized a 5-min warmup period at 65 watts (W); intensity was then set to 65 W below the predetermined peak power output and was increased by 33 W every 2 min until the subject could no longer maintain 50 revolutions per minute (rpm). A 60% power output was then predicted and used for the experimental sessions. Subjects were asked to record their dietary intake and physical activity during the three days prior to the first experimental procedure. This recorded pattern was then repeated prior to the subsequent session. The order of treatment was counterbalanced alternately among the subjects, and a minimum of

one week elapsed between experiments. During the 24 hr prior to exercise, fluid and food intake were maintained at equal levels in both protocols and all urine was collected.

Protocol– Subjects entered the Human Research Facility at the Ames Research Center at 0900 the day prior to the exercise testing. In HDT24, after check-in procedures, 25 hr prior to exercise the subject began 1 hr of upright chair rest at 1000 hr. A pretilt blood sample was taken, and then at 1100 hr the subject was placed in HDT bed rest for 24 hr. At 1000 hr on the second day the subject was prepared for exercise. Additional blood samples were obtained at HDT24 (preexercise), during exercise at min 10, 20, and 68, and 10 min after exercise. Oxygen consumption was measured continuously during exercise. The T_{re}, $\overline{T}_{sk}$, sweat rate, and skin blood velocity (SBV) were measured before exercise and every 10 min during exercise; heart rate was measured every 10 min during exercise. There was no forced ambient air flow during exercise. The procedure for HDT1 was similar to that for HDT24 except that the subject remained ambulatory during the 22 hr prior to exercise except for 8 hr of sleep in a horizontal posture from 2300 hr to 0700 hr. The pretilt blood sample was obtained after 1 hr chair rest prior to HDT1; the preexercise sample was obtained after HDT1. All blood samples were obtained at the same time of day in both HDT1 and HDT24.

Procedures– An 18-gauge Teflon catheter (Quik-cath, Travenol Laboratories Inc., Deerfield, IL 60015) was inserted in a large vein in the antecubital space of the forearm for blood sampling. Each blood sample was 7 milliliters (ml). The volume of blood comprising the dead space was discarded prior to sampling, and the catheter was maintained patent with a sodium heparin flush following each sample. To obtain Hct, quadruplicate microcapillary tubes were spun for 5 min at 11,500 rpm on an International Microcapillary Centrifuge, model MB; they were read on an International Microcapillary Reader, model CR (International Equipment Co., Needham Heights, MA 02194). Raw Hct values were multiplied by 0.91 to correct from venous to whole body Hct. The [Hb] was measured in triplicate on a Coulter Hemoglobinometer (Coulter Electronics, Hialeah, FL, 33010). Changes in PV relative to pretilt values were calculated from Hct and [Hb] (Greenleaf et al., 1979). The plasma osmolality was measured by freezing point depression on an Advanced Digimatic Osmometer, model 3D-II (Advanced Instruments Inc., Needham Heights, MA 02194). Plasma samples were also analyzed for sodium ($[Na^+]$) and potassium ($[K^+]$) on a Beckman System E2A Electrolyte Analyzer (Beckman Instruments, Inc., Brea, CA 92621), and plasma was analyzed for total protein and albumin concentrations with a Cobas Mira analyzer (Roche Diagnostic Systems, Nutley, NJ 07110).

The metabolic gas collection system utilized indirect calorimetry and computerized data acquisition (Greenleaf et al., 1989). Heart rate data were collected on a Hewlett Packard cardiotachometer model 78905A and ECG module model 78203C (Hewlett Packard, Medical Products Group, Waltham, MA 02154).

Sweat rate was determined at three sites before exercise and every 10 min thereafter by using resistance hygrometry (Bullard, 1962) (Hygrometery sensors, Thunder Scientific, Albuquerque, NM 87123). The sweat capsules were placed on the lateral surfaces of the midregion of the upper arm, midthigh, and on the belly of the gastrocnemius. Data collection and calculations of relative humidity for the sweat rate calculations were performed by a Digitec Data Scan Sentinel 1100 (United Systems Corp., Dayton, OH 45401).

A laser Doppler system (Laser Flo model BPM 403A, TSI Inc., St. Paul, MN 55164) was used to estimate regional blood perfusion. The SBV in Hertz (Hz) $\times 10^2$ was measured with the Doppler probe secured at the temple. The percent relative change from preexercise values were calculated and compared.

Yellow Springs Instruments series 700 thermistors (Yellow Springs, OH 45387) were used to measure $\overline{T}_{sk}$ and T_{re}: $\overline{T}_{sk}$ was measured at seven sites and calculated with weighting as follows (Greenleaf and Castle, 1971):

$$\overline{T}_{sk} = \text{right chest } (0.19) + \text{left chest } (0.20) + \text{upper arm } (0.06) + \text{forearm } (0.13) + \text{calf } (0.21) + \text{thigh } (0.21)$$

The T_{re} was measured using a flexible nylon thermistor probe inserted to a depth of 12 cm. All temperatures were recorded on a Squirrel (model SQ32-2YS/8YS/1V/HR) type data logger (Science/Electronics Inc., Miamisburg, OH 45342).

The rh was calculated from a Bendix model 566 psychrometer (Bendix Environmental Science Div., Baltimore, MD 21204).

Statistical methods– Statistical analysis was performed with the General Linear Models Procedure (SAS Institute Inc., Cary, NC). The design was a repeated measures analysis of variance (ANOVA) utilizing no grouping factors and two within factors; two treatments and time points within a treatment. Contrast decompositions were used to compare further the effect of treatment, time, and time by treatment interactions. Post hoc comparisons were preselected for mean cell differences of interest, and a Bonferroni correction was made for the

number of comparisons within an analysis. Variables confirming the presence of a volemic treatment effect (Hct, [Hb], PV) were evaluated using a two-tailed test. If the direction of an observed treatment effect on a variable reflected a preconceived effect due to volemic state, a one-tailed test was used to evaluate treatment effects. Values of $p < 0.05$ were used to reject the null hypothesis. Results are expressed as mean ± standard error unless otherwise stated.

Results

Blood variables– The results of the blood-sample analyses are presented in table 3-1. Whereas all the pretilt values were not different between HDT1 and HDT24 (NS, $p < 0.26$), the preexercise (0 min) values display a significant decrease in Hct from 43.5% to 42.2% in HDT1 ($p < 0.003$) and an increase from 42.4% to 44.3% in HDT24 ($p < 0.01$). When the ANOVA is performed with the preexercise values as the contrast variable, there is no time-treatment interaction ($p < 0.10$); therefore, the preexercise treatments caused no difference in the exercise response between HDT1 and HDT24. The Hct at 10 min after exercise remained elevated with reference to preexercise for both HDT1 and HDT24. The [Hb] decreased significantly ($p < 0.004$) from 16.2 to 15.6 $g \cdot 100\ ml^{-1}$ with HDT1, but a change from 16.4 to 16.7 $g \cdot 100\ ml^{-1}$ after HDT24 was not significant ($p < 0.50$). The results of the ANOVA for [Hb] showed a nonsignificant time-treatment interaction (NS, $p < 0.16$) but a significant effect for both treatment ($p < 0.007$) and time ($p < 0.0001$). Post-exercise values for [Hb] remained elevated above preexercise values for HDT1 and HDT24. Figure 3-1 displays the PV changes calculated from pretilt chair rest Hct and [Hb] values. Changes in PV showed a significant effect of treatment ($p < 0.04$), time ($p < 0.0001$), and treatment-time interaction ($p < 0.01$). In the treatment-time decomposition, the only significant difference existed in the baseline to preexercise change ($p < 0.03$), where PV increased 6.1% with HDT1 and decreased 4.3% with HDT24. During exercise, the PV decreased 6.7% at min 10 and reached a nadir of –8.1% at min 60 in HDT1. In HDT24, the PV decreased 12.7% at min 10 with a nadir of –15.3% at min 60. The plasma osmolality was unchanged by both treatments (288 ± 1 m to 287 ± 1 mosm/kg for HDT1, and 287 ± 1 to 288 ± 1 mosm/kg for HDT24 (NS, $p = 1.00$). The plasma osmolality increased significantly during exercise and remained elevated 10 min after exercise. Plasma [Na^+] did not change with preexercise treatment, but it increased significantly during exercise ($p < 0.0001$) and remained elevated 10 min after exercise. Total protein was significantly reduced with HDT1, decreasing from 6.8 ± 0.1 to 6.6 ± 0.1 $g \cdot 100\ ml^{-1}$ ($p < 0.04$). Values increased significantly with exercise ($p < 0.0001$) and remained elevated 10 min after exercise in both conditions. Plasma albumin showed no change with treatment ($p < 0.74$), but it increased significantly ($p < 0.001$) during and following exercise in both conditions.

Fluid balance– With similar environmental conditions and dietary and fluid intakes imposed in the two preexercise time periods, the differences in 24-hr urine output were attributed to the ambulatory or HDT conditions preceding exercise. Total output was 2143 ± 162 ml (1.49 $ml \cdot min^{-1}$) for HDT1 and 2740 ± 294 ml (1.90 $ml \cdot min^{-1}$) for HDT24 ($p < 0.05$).

Temperatures– Figure 3-2 displays the T_{re} changes before and during exercise. The preexercise tilt effect on T_{re} during exercise was not significant ($p < 0.07$). Post hoc analysis revealed a significant difference in T_{re} prior to exercise with HDT1 at 36.71°C ± 0.06 and with HDT24 at 36.93°C ± 0.11 ($p < 0.04$); the increases of 1.30°C in HDT1 and 1.33°C in HDT24 after exercise were significant ($p < 0.0001$). The average $\overline{T}_{sk}$ was higher in HDT24 than in HDT1 until min 40 (fig. 3-3). The increase during exercise was significant ($p < 0.0001$). A significant time-treatment interaction occurred in HDT24 ($p < 0.003$) as $\overline{T}_{sk}$ leveled off at 31.1°C between min 30 and 40, whereas in HDT1 $\overline{T}_{sk}$ continued to increase to 31.5°C at min 70.

Sweat rates– Total weight loss during exercise was not different ($p < 0.15$) between HDT1 (1.63 ± 0.24 kg) and HDT24 (1.33 ± 0.09 kg). Sweat rates measured at three sites had significant time effects ($p < 0.001$) but no significant differences between HDT1 and HDT24. Sweat rates for HDT1 and HDT24, respectively, at min 60 reached levels of 0.301 ± 0.043 $g \cdot cm^{-2} \cdot hr^{-1}$ and 0.285 ± 0.032 $g \cdot cm^{-2} \cdot hr^{-1}$ for the forearm ($p < 0.30$), 0.453 ± 0.044 $g \cdot cm^{-2} \cdot hr^{-1}$ and 0.485 ± 0.060 $g \cdot cm^{-2} \cdot hr^{-1}$ for the upper arm ($p < 0.16$), and 0.418 ± 0.036 $g \cdot cm^{-2} \cdot hr^{-1}$ and 0.506 ± 0.087 $g \cdot cm^{-2} \cdot hr^{-1}$ for the calf ($p < 0.18$).

Laser Doppler skin blood velocity– Percent changes in SBV ($Hz \times 10^2$) were calculated from preexercise values (fig. 3-4). The time effect ($p < 0.0001$) was significant, but the treatment ($p < 0.16$) and time-treatment interaction ($p < 0.39$) effects were not. The percent increases over preexercise at min 70 were 270 ± 60% for HDT1 and 210 ± 70% for HDT24.

Heart rate– The increases in heart rate during exercise from 123 ± 4 bpm to 144 ± 4 bpm in HDT1 and from 126 ± 4 bpm to 155 ± 5 bpm in HDT24 (fig. 3-5) indicated that the time effect ($p < 0.0001$) was significant, but the treatment ($p < 0.11$) and time-treatment interaction ($p < 0.37$) had no effect.

Table 3-1. Plasma constituents

Treatment	Supine exercise (min)					
	Pretilt	0	10	20	68	Post
		Hct (%)				
HDT1	43.5[a]	42.2[a,b]	44.8[c]	44.7[c]	44.6[c]	43.0[c]
	(0.8)	(0.8)	(0.7)	(0.8)	(0.7)	(0.8)
HDT24	42.4[a]	44.3[a,b]	46.2[c]	46.5[c]	46.4[c]	45.6[c]
	(1.0)	(0.8)	(0.9)	(1.2)	(0.8)	(0.9)
		[Hb] ($g \cdot 100\ ml^{-1}$)				
HDT1	16.2	15.6[b]	17.0[c]	16.9[c]	17.3[c]	16.2[c]
	(0.4)	(0.3)	(0.3)	(0.3)	(0.4)	(0.2)
HDT24	16.4	16.7	17.7[c]	17.9[c]	18.1[c]	17.3[c]
	(0.2)	(0.2)	(0.3)	(0.4)	(0.3)	(0.3)
		Osmolality (milliosmols $\cdot$ kg H_2O^{-1})				
HDT1	288	287	292[c]	292[c]	294[c]	289[c]
	(1)	(1)	(1)	(1)	(1)	(1)
HDT24	287	288	291[c]	292[c]	294[c]	290[c]
	(1)	(1)	(1)	(1)	(1)	(1)
		[Na^+] (millimols $\cdot$ l^{-1})				
HDT1	143	143	146[c]	146[c]	147[c]	144[c]
	(1)	(1)	(0)	(0)	(1)	(0)
HDT24	143	143	146[c]	146[c]	147[c]	145[c]
	(1)	(0)	(0)	(0)	(0)	(0)
		Total protein ($g \cdot 100\ ml^{-1}$)				
HDT1	6.8	6.6[b]	7.2[c]	7.3[c]	7.3[c]	7.0[c]
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)
HDT24	6.8	6.8	7.3[c]	7.2[c]	7.5[c]	7.2[c]
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)
		Albumin ($g \cdot 100\ ml^{-1}$)				
HDT1	4.4	4.4	4.8[c]	4.8[c]	4.8[c]	4.6[c]
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)
HDT24	4.4	4.4	4.7[c]	4.8[c]	4.9	4.7[c]
	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)	(0.1)

[a]Denotes a significant time-treatment interaction between HDT1 and HDT24 pretilt to time 0 ($p < 0.05$).
[b]Denotes a significant difference from pretilt to time 0 (preexercise) ($p < 0.05$).
[c]Denotes a significant difference from time 0 ($p < 0.05$).

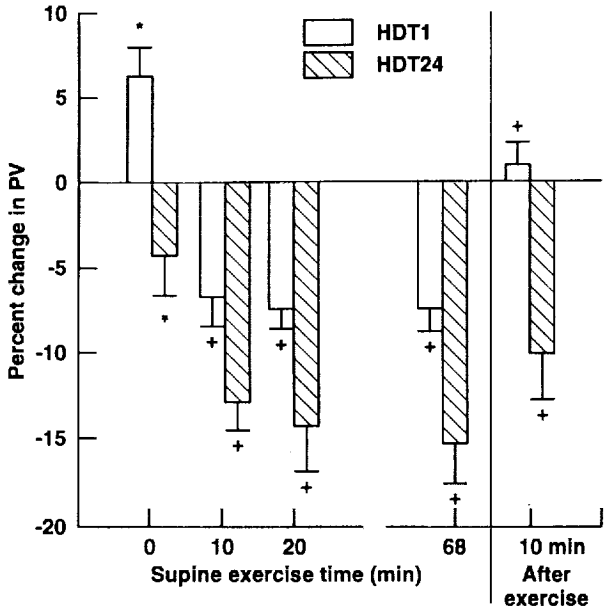

Figure 3-1. Percent change in PV calculated before and during exercise. Asterisk denotes significant effect of HDT ($p < 0.05$). Plus sign denotes significant time effect from preexercise ($p < 0.05$). The time-treatment interaction was significant at $p < 0.03$.

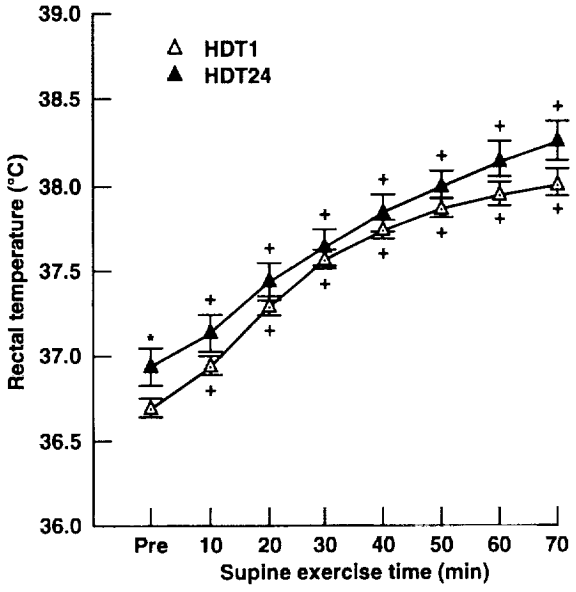

Figure 3-2. Change in T_{re} before and during exercise. Symbols are means ±SE ($n = 7$). Asterisk denotes a significant elevation above HDT1 preexercise ($p < 0.05$). Plus sign denotes significantly elevated above respective preexercise datum ($p < 0.05$).

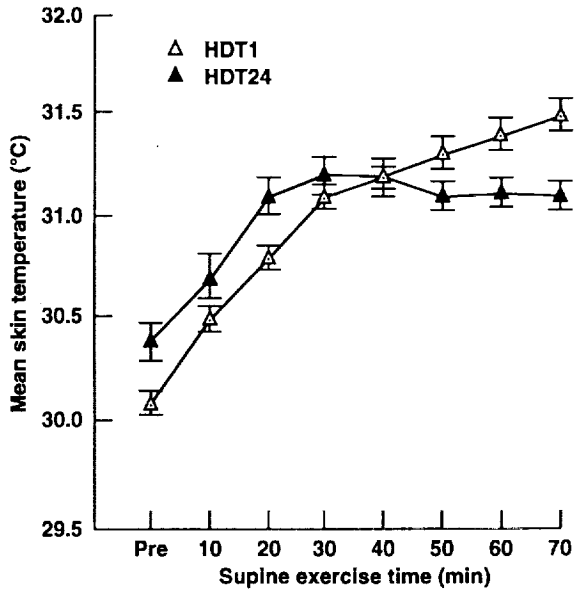

Figure 3-3. Change in $\bar{T}_{sk}$ before and during exercise. The time-treatment interaction was significant at $p < 0.003$.

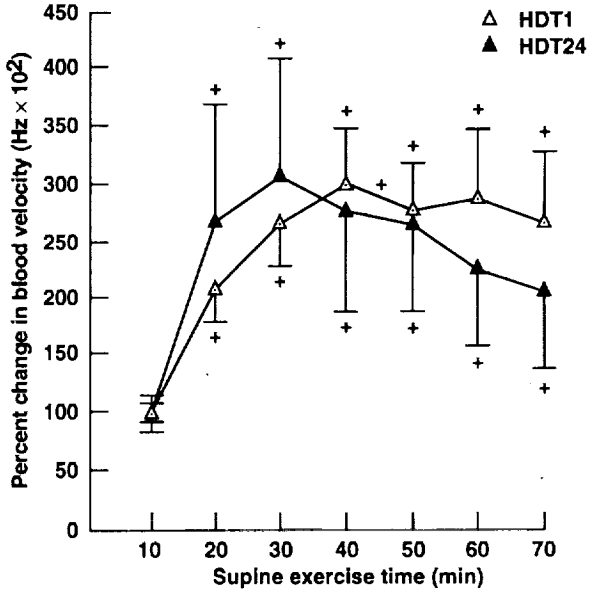

Figure 3-4. Percent change in SBV measured by laser Doppler during exercise. Asterisk denotes a significant difference ($p < 0.05$) from min 10 value. There was no significant treatment or time-treatment effect.

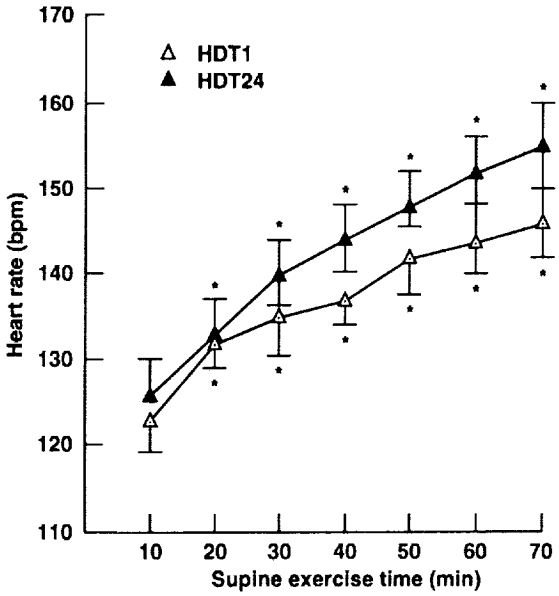

Figure 3-5. Heart rate during exercise. Asterisk denotes a significant difference ($p < 0.05$) from min 10 value. There were no differences between treatments.

Discussion

The hypothesis proposed that HDT24 and an associated reduction in PV would result in higher exercise T_{re} compared to exercise T_{re} for HDT1 and its associated increase in PV. The preexercise changes in PV were significant and in the direction expected. When compared to upright chair rest data, HDT1 caused a 6.1% increase in PV. A range of PV from 11% to 16% between supine and upright postures has been reported, with approximately a 10% difference between sitting and standing (Hagan et al., 1978; Harrison, 1985). When seated chair rest was used as a reference baseline, 10° HDT resulted in progressive increases in PV that reached 14.5% at 5 hr (Gharib et al., 1988). The effect on relative change in PV seen in the present study using HDT1 is within an expected range for this change in posture. A seated upright reference posture has been suggested to be the control position with which to evaluate changes caused by HDT (Gharib et al., 1988). An upright posture when followed by HDT elicits greater fluid changes, which would be attenuated when compared to the already-fluid-shifted supine posture. Consequent to the redistribution of body fluids and the increase in PV with HDT, HDT24 caused a 4.3% reduction in PV relative to the pretilt baseline. Although there was no clear association between PV and water balance during HDT bed rest, the relative 600 ml diuresis and net reduction of 10.4% of PV with HDT24, when compared to the 24-hr ambulatory condition preceding HDT1, is comparable to the 776-ml HDT 24-hr negative fluid balance and approximate 11% decrease in PV measured after 8 days of 6-deg HDT in five subjects (Greenleaf et al., 1992).

Acute decreases in core temperature occur in several conditions in which an upright posture is compared to a supine posture where hydrostatic gradients are reduced, or 12° HDT (Asmussen et al., 1940; Kleitman and Dokortsky, 1933; Novak et al., 1988). In the present study preexercise T_{re} was significantly lower after HDT1 than after HDT24. The preexercise T_{re} may have been influenced by the change in posture from the pretilt chair rest reference posture and the reduction in PV with HDT24. Upright chair rest was used as a reference to elicit significant adjustments to the HDT posture. The overall change in hydrostatic gradients with a change in posture from upright to supine stimulates baroreceptors to reduce sympathetic outflow and allow SkBF to rise, resulting in an increased conductive heat loss and a lower core temperature (Rowell, 1986). It is possible that the increase in central BV and PV during HDT1 resulted in a lowering of T_{re}, whereas the decrease in PV with HDT24 removed some baroreceptor input that reduced sympathetic vasoconstriction of the cutaneous vasculature. Subsequently, T_{re} was higher after 24 hr. Pannier et al. (1991) reported a decrease in forearm vascular resistance up to 6-hr HDT, which returned to baseline values after 24 hr. A decrease in PV was implicated as mediating this response, although PV changes were not reported.

The increases in T_{re} during exercise were similar in HDT1 and HDT24 (no time treatment interaction). The endogenous heat production during 70 min of submaximal exercise was used rather than an ambient heat stress to activate thermoregulatory mechanisms. This exercise regimen in comparable moderate ambient conditions has resulted in significant increased core temperatures following heat and exercise dehydration with cycling at 49% of $\dot{V}O_{2\,max}$ (Greenleaf and Castle, 1971), and following 14 days of bed rest with cycling at 43% to 48% of $\dot{V}O_{2\,max}$ (Greenleaf and Reese, 1980).

Initial HDT24 $\overline{T}_{sk}$ during exercise was higher than HDT1 $\overline{T}_{sk}$; those in HDT24 exercise leveled off between 30 and 40 min of exercise, whereas those in HDT1 exercise exceeded those in HDT24 at min 40 and continued to increase throughout exercise. Lower $\overline{T}_{sk}$ and calculated skin heat conductance accompanying higher core temperatures occur during exercise with PV reduced following the use of diuretics (Claremont et al., 1976) and 14 days of bed rest (Greenleaf and Reese, 1980). The reduced $\overline{T}_{sk}$ and skin conductance suggest inhibited heat transfer from the core to the periphery with lower $\overline{T}_{sk}$ reflecting a decrease in heat loss in similar environmental

temperatures. Skin blood velocity in HDT1 remained relatively constant at 270% to 300% between min 30 and 40 of exercise, whereas in HDT24 the increase was 310% at min 30 and decreased to 210% at min 70. The mean decreasing trend (NS) of SBV displayed a similar trend to that seen in $\overline{T}_{sk}$. The reduced $\overline{T}_{sk}$ and SBV in HDT24 suggest a decrease in peripheral circulation as exercise continued beyond 40 min. Exercise had no significant effect on T_{re} possibly because of the magnitude of the peripheral changes and the timing of the inferred leveling off or decrease in peripheral circulation. The elevated preexercise T_{re} in the present study would cause threshold T_{re} for thermoregulatory increases in SkBF to be reached earlier. In contrast, the reduction in PV with HDT24 increases the threshold values for thermoregulation behavior; this interaction resulted in no difference in the increases of T_{re} with exercise.

The sweat rate measured over both active and inactive muscles in the present study showed no significant differences between treatments or with a time-treatment interaction. No significant changes occurred in plasma osmolality or $[Na^+]$ between HDT1 and HDT24 to affect the sweating responses. Osmotic changes in the plasma can affect the threshold core temperature for onset of sweating. Hyperosmolality without reduction in PV delays the onset of sweating and results in a higher core temperature during exercise (Nielsen, 1974, Fortney et al., 1984). Hypovolemia alone affects sweat rate (Fortney et al., 1981). A 15.9% isosmotic reduction in PV induced by diuretics reduced the slope of the esophageal-to-sweat-rate relationship measured over inactive muscle when exercise intensity was 65% to 70% of $\dot{V}O_{2\,max}$. The combination of the level of hypovolemia, exercise intensity, and the lack of ambient heat stress may have been of insufficient severity to elicit significant changes in sweating in the present study.

The changes in PV during exercise in the present study were significantly different only as a result of the preexercise treatments. The relative hypovolemic changes due to exercise at min 68 were 13.4% with HDT1 and 11.4% with HDT24. The slight dilution of total plasma proteins and the increase in preexercise PV in HDT1 may have had a small but insignificant effect of allowing a greater hemoconcentration with exercise. Exercise causes loss of plasma water from the vascular space (hemoconcentration) that is dependent both on preexercise posture and exercise intensity (Diaz et al., 1979; Hagan et al., 1980).There is an upper limit for exercise hemoconcentration dependent on the overall balance of Starling forces across the microcirculatory vessels (Fortney et al., 1981; Harrison, 1985). A greater loss of fluid prior to exercise in the present study would have concentrated plasma proteins to a greater degree than observed, but the vascular transcapillary pressures may have been lower in a hypovolemic state and this condition would have a balancing effect on the Starling forces.

Increases in heart rate during exercise reflect thermoregulatory and hydrostatic shunting of blood to the periphery (Rowell, 1986). Heart rate during exercise showed a progressive increase that has been termed cardiac drift (Rowell, 1986). The decrease in $\overline{T}_{sk}$ and the trend for decreased SBV suggests that peripheral vasoconstriction preserved central BV and prevented increases in heart rate from being significantly different between HDT24 and HDT1.

The hypothesis of an increased T_{re} during exercise following HDT24 was not affirmed. The significantly elevated preexercise T_{re} may have caused threshold T_{re} for thermoregulatory increases in SkBF and sweating to be reached earlier. In contrast, reductions in PV increase the threshold values for thermoregulatory behavior. Whereas the $\overline{T}_{sk}$ and SBV responses inferred an attenuation of SkBF during exercise after HDT24, the time-treatment interaction of the increase in T_{re} during exercise was not different between HDT1 and HDT24. The interplay of the elevated preexercise T_{re} and the reduced PV prior to exercise may have offsetting influences on threshold levels for thermoregulatory behavior.

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4.0 Shifts in Plasma Volume with Water Immersion and HDT

Summary

The purpose of this study was to determine if the transient increase and subsequent decrease in PV with 6-deg HDT could be reproduced with varied periods of up to 6 hr of WI. The compared treatments were HDT1, HDT24, and 34.5°C WI. All were preceded by 1 hr of upright chair rest beginning at 1000 hr in HDT and 0800 hr in WI. Changes in PV, calculated from chair rest Hct and [Hb], were monitored hourly during WI to determine relative changes in PV. With HDT1 and following 1 hr HDT in HDT24, PV had significantly increased by 6.1 ± SE 0.9% and by 6.8 ± 1.5% ($p < 0.05$), respectively. After 1 hr of WI, PV had increased by 7.7 ± 2.1% ($p < 0.05$); there was no significant difference between treatments at 1 hr. Following HDT24 and WI, PV had been reduced by 4.3 ± 2.3% and by 1.1 ± 1.8% ($p < 0.05$), respectively. Although the reductions in PV were not significantly different, great intraindividual variability occurred in the response to HDT24 and WI. In a comparison between the change in PV from chair rest and the response to HDT24 or WI, the difference in PV was 4.2 ± 2.8%. The mean time for WI was 4.4 ± 1.2 hr (median = 4 hr). Two subjects remained in WI for 6 hr without attaining predetermined decreases in PV. The differences in PV in these subjects were 14.9% and 8.7% between HDT24 and WI. In another subject PV decreased 8.9% more than HDT24 after 3 hr of WI. The ability to reproduce PV changes consistently with HDT and WI is limited by intraindividual variability.

Introduction

The transient increase and subsequent decrease in PV is a salient and reproducible feature of both HDT and WI. The reference posture prior to HDT and WI has a major influence on the PV change. In both cases an upright seated posture has been suggested to be an appropriate reference to measure significant changes in PV, the cardiovascular system, and the endocrine system (Gharib et al., 1988; Epstein et al., 1989). With the postural change of HDT, the effect of gravitationally induced hydrostatic gradients is reduced by altering the vector along the axis of the body. In WI the hydrostatic counterpressure causes compression of tissues. In both cases there is a headward shift of body fluids to central reservoirs and an alteration in the balance of Starling forces across exchange vessels, and these conditions result in a transient increase in PV (Blomqvist and Stone, 1983). The effect of WI on PV has been suggested to be a special case of a postural hemodilution (Harrison et al., 1986). The time course for HDT or WI hemodilution is comparable to that of a change from upright to supine posture, where peak increases are reached within an hr (Hagan et al., 1978). Consequent to the increase in central BV and PV, a volume regulating diuresis and natriuresis will reduce PV over time.

The actual decrease in PV with HDT24 and the time required to decrease PV with WI to that same level was previously unknown. Since both methods are used to simulate the physiological responses to microgravity, the validity of cross comparisons becomes an issue of some importance. Therefore, the duration of WI was varied to elicit comparable reductions in PV.

Methods

A minimum of one week elapsed between the HDT and the WI experiments. An effect of treatment order existed, but the HDT24 treatment had to be performed prior to WI to determine the goal PV change during WI. Prior to WI the subjects remained ambulatory; they slept in a horizontal posture from 2300 hr to 0700 hr. On the day of WI the subject began 1 hr of upright chair rest at 0800 hr. During chair rest a venous catheter was inserted as described in Chapter 3. At 0900 hr a 7-ml preimmersion blood sample was drawn for baseline measures of Hct and [Hb] concentration. The subject then began 3 to 6 hr of WI to the neck in 34.5°C water. At hourly intervals a 1-ml blood sample was drawn and analyzed immediately to calculate relative changes in PV. If the relative change in PV approached the PV change of HDT24, another sample was taken on the half hr. If the target PV change was achieved before 6-hr WI, the subject left the tank and assumed a supine posture on a gurney. The maximum allotted time for WI was set at 6 hr; a final blood sample was taken 30 min after WI. Statistical analysis was performed with the General Linear Models Procedure (SAS Institute Inc., Cary, NC). The design was a repeated measures ANOVA and utilized no grouping factors and two within factors (two treatments and time points within a treatment). Contrast decompositions were used to further compare the effect of treatment, time, and time-by-treatment interactions. Post hoc comparisons were preselected for mean cell differences of interest, and a Bonferroni correction was made for the number of comparisons within an analysis. Values of $p < 0.05$ were used to reject the null hypothesis. Results are expressed as mean ± SE. Several statistical comparisons were of particular interest: chair rest values between treatments and comparisons between chair rest and HDT1, HDT24, 1-hr WI, after HDT24, and after WI.

Results

Figures 4-1 and 4-2 present Hct and [Hb] values, respectively. An early morning hemodilution was observed; chair rest Hct for pre-WI (41.4 ± 0.8) was significantly lower than Hct for pre-HDT1 (43.5 ± 0.8) ($p < 0.05$). In addition, mean values for pre-WI were lower than mean values for HDT24 (42.4 ± 1.0). At both HDT1 and WI all Hct and [Hb] values were significantly lower than those for chair rest ($p < 0.05$). After HDT24, values of Hct and [Hb] were significantly higher than those for HDT1. At the end of WI, Hct and [Hb] values were not different from those measured at HDT1. Figure 4-3 displays the percent PV changes. After HDT1 and 1 hr in HDT24, the PV had increased significantly by 6.1 ± SE 0.9% and 6.8 ± 1.5% ($p < 0.05$), respectively. After 1-hr WI, PV had increased by 7.7 ± 2.1% above chair rest PV ($p < 0.05$). There was no significant difference in PV between treatments at 1 hr, but PV was reduced by 4.3 ± 2.3% after HDT24 and by 1.1 ± 1.8% after WI ($p < 0.05$). The reductions in PV were not significantly different between HDT24 and WI. The individual subject changes in PV along with WI duration for HDT24 and WI are presented in table 4–1. The average percent PV difference between post HDT24 and WI was –4.2 ± 2.8%; the average time in WI was 4.4 ± 1.2 hr. Two subjects remained in WI for the entire 6 hr with differences of –14.9% and –8.7% between HDT24 and WI. Another subject had a +8.9% difference after 3 hr of WI.

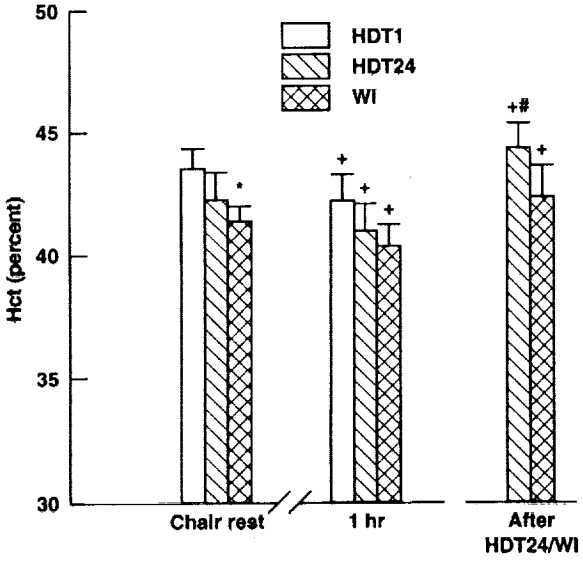

Figure 4-1. Values of Hct for chair rest, HDT1, HDT24, and WI. Asterisk denotes values of Hct significantly different from HDT1 at a given time point ($p < 0.05$). Plus sign denotes change within treatment from chair rest ($p < 0.05$). Pound sign denotes values significantly different from HDT1 1-hr value ($p < 0.05$).

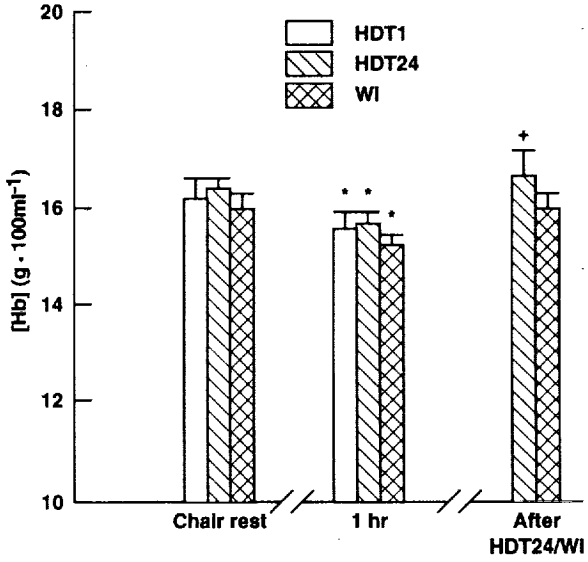

Figure 4-2. Values of [Hb] for chair rest, HDT1, HDT24, and WI. Asterisk denotes significantly different [Hb] than chair rest ($p < 0.05$). Plus sign denotes significantly different from HDT1 1-hr treatment ($p < 0.05$).

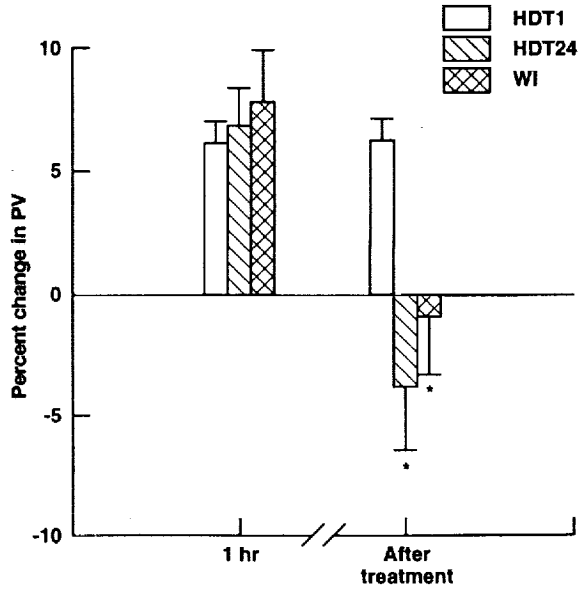

Figure 4-3. Changes in PV calculated from pretreatment chair rest at 1 hr and after treatment. Asterisk denotes values of PV significantly different from PV for HDT1 ($p < 0.05$).

Discussion

The relative decrease in PV associated with HDT24 was statistically reproduced with a WI of 3 to 6 hr, but the time of day and the subject variability affected the

Table 4-1. Comparison of percent changes in PV after HDT24 and 3 to 6 hr of WI. Calculated from pretreatment chair rest Hct and [Hb] values

Subject	Treatment	PV (%) change	(HDT24% – WI%)	WI time (hr)
243	HDT24	2.4		
	WI	6.1	–3.7	3.0
292	HDT24	3.8		
	WI	–5.1	+8.9	3.0
385	HDT24	–1.2		
	WI	–1.5	+0.3	4.5
440	HDT24	–6.6		
	WI	1.1	–7.7	4.0
441	HDT24	–13.8		
	WI	1.1	–14.9	6.0
442	HDT24	–10.7		
	WI	–7.1	–3.6	4.0
443	HDT24	–4.3		
	WI	4.4	–8.7	6.0

baseline chair rest values of Hct and [Hb]. Subjects began the preimmersion baseline chair rest at 0800 hr so that the timing of the end of WI would coincide with the end of 24-hr HDT. The value of Hct for the preimmersion blood sample was significantly lower for HDT1 than for WI. The HDT24 chair rest value was intermediate, possibly reflecting subject variability between HDT1 chair rest and the time of day that the chair rest blood sample was taken.

Although some subjects met the projected comparable decreases in PV, others spent an entire 6-hr period of WI without reaching the preselected changes. The pretreatment posture had a significant effect: when a supine reference posture is used, the effects of HDT and WI are lesser in magnitude. In the present study a significant increase in PV of 6% to 8% occurred at HDT1 and WI. The subsequent reduction in PV with HDT24 was approximately 10% when compared to the HDT1 values. Nixon et al. (1979) used a supine reference and 24 hr of 5-deg HDT to reduce PV by 11%. Gharib et al. (1988) utilized seated chair rest as reference and found a peak PV increase of 14.5% within an hr, but after 5 hr the hypervolemia was reduced to approximately 7%. Evidently the hypervolemic response to HDT occurs regardless of reference posture used, but the level of transient change will vary according to the reference posture. Chronic PV changes with HDT or WI will be of the same magnitude if it is assumed that those occurring in a supine posture are in a transient stage of hypervolemia.

One-hr WI in the present study caused a significant relative increase in PV of 7.7%, the same magnitude as HDT1 with the same reference posture. The subsequent reduction varied greatly: one subject experienced a decrease of 8.9% beyond the HDT24 reduction in PV after only 3 hr, whereas two subjects failed to meet the reduction in PV even after 6 hr of WI. The reasons for this variability between subjects are unknown. McCally (1965) was the first to document the transient increase and ensuing reduction in PV with WI. With no reference to preimmersion posture, McCally's subjects, allowed to assume any comfortable position during WI, experienced a peak increase in PV of 9% within 30 min and a reversal within 1 hr. After 6 hr PV was reduced by 11%. When a supine reference is used and the subjects are hydrated during immersion, PV increases nonsignificantly, but after 8 hr it is reduced by nearly 16% (Greenleaf et al., 1981). If a standing preimmersion reference posture is used the transient increase in PV can reach 15.5% after 75 min; in addition, the increase with a standing reference is maintained longer than with a supine reference (Harrison et al., 1987).

In both HDT and WI the magnitude of the transient increase and subsequent decrease in PV depends on the pretreatment reference. Both HDT and WI caused similar increases in PV at 1 hr. Perhaps the transient hemodilution of HDT or WI would reduce variability in the PV response if the hemodilution were used as the reference with which to compare subsequent reductions in PV.

The changes in PV with HDT and WI were difficult to predict and to control. Time of day affected the pre-WI Hct and [Hb] and the intraindividual and interindividual responses to the treatments. Six-hr WI was not always adequate time to elicit comparable changes to HDT24 PV levels. Individual variation in responses may prevent

direct comparisons of HDT24 and WI. Evaluation of the suitability of these two models in simulating PV changes with weightlessness needs to account for the differences in the overall response and its reproducibility. It seems appropriate to do repeated measures of WI responses on given individuals: subjects with large hypervolemic responses; subjects whose PV declines rapidly; subjects who respond slowly; etc. Selected physiological profiles might develop on subjects who have characteristic responses. Insight could then be gained on mechanisms involved in the varied responses. Other questions to be investigated are: Would transient PV changes be similar with HDT and WI when reference blood values are comparable? and How can daily variations be controlled?

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5.0 Conclusion

The PV increase with HDT1 and the PV decrease with HDT24 are associated with a change in rectal temperature at rest. The rectal temperature was significantly elevated after HDT24. Plasma volume changes of this magnitude will influence sympathetic outflow to the cutaneous circulation. A decrease in PV may cause cardiopulmonary reflexes to effect a relative cutaneous vasoconstriction resulting in reduced heat loss. This mechanism could result in the observed increase in rectal temperature. The $\overline{T}_{sk}$ and SBV, factors associated with cutaneous circulation, were reduced during exercise after HDT24. After HDT24 the resting T_{re} was elevated bringing core temperature closer to threshold values to increase cutaneous blood flow. On the other hand PV was reduced resulting in a possible increase in the threshold for cutaneous vasodilation. During exercise the increases in T_{re} were parallel after HDT1 and HDT24 suggesting the probability of offsetting effects of preexercise increased T_{re} and reduced PV. The level of hypovolemia, the intensity of exercise, the supine posture during exercise, and the level of ambient heat stress may not have been of sufficient severity to elicit significant changes in sweating. These levels of PV change affect body temperatures at rest and during exercise. These findings emphasize the importance of PV maintenance during HDT bed rest deconditioning and spaceflight to enhance exercise thermoregulation and work capacity.

APPENDIX

DATA

Hematocrit × 0.91

HDT1			Minutes				
Sub. #	C.R.[a]	1 hr	0	10	20	68	Rec[b]
243	45.7	43.9	43.9	45.7	46.2	46.2	44.5
292	44.2	43.6	43.6	46.8	46.6	45.4	44.2
385	39.5	37.5	37.5	40.6	39.9	41.0	39.1
440	44.0	41.9	41.9	44.9	45.0	44.7	42.8
441	44.6	42.9	42.9	45.5	45.3	45.9	44.2
442	45.2	44.4	44.4	46.4	46.3	46.0	44.9
443	41.4	41.1	41.1	43.6	43.2	43.2	41.0
Mean	43.5	42.2	42.2	44.8	44.7	44.6	43.0
STD	2.1	2.2	2.2	2.0	2.2	1.7	2.0
SE	0.8	0.8	0.8	0.7	0.8	0.7	0.8

HDT24								
Sub. #	C.R.	1 hr	24 hr	0	10	20	68	Rec
243	44.2	42.0	44.4	44.2	46.7	46.9	47.6	46.3
292	44.1	41.7	43.8	45.0	46.1	46.2	46.0	45.0
385	39.8	38.2	40.4	42.7	44.1	44.1	43.9	43.2
440	44.4	43.5	45.0	45.9	47.8	51.5	48.2	47.7
441	38.8	38.0	42.0	42.9	45.0	44.8	46.3	45.6
442	46.2	45.9	48.7	48.1	50.4	50.2	49.7	49.2
443	39.5	39.0	40.4	41.0	43.1	42.0	43.1	42.0
Mean	42.4	41.2	43.5	44.3	46.2	46.5	46.4	45.6
STD	2.8	2.7	2.7	2.2	2.3	3.1	2.2	2.3
SE	1.0	1.0	1.0	0.8	0.9	1.2	0.8	0.9

WI				
Sub. #	C.R.	1 hr	End	Post
243	42.4	41.8	43.0	43.0
292	42.2	41.5	43.9	42.8
385	38.0	35.7	36.9	37.3
440	42.4	42.2	44.3	44.6
441	42.2	41.0	42.7	43.6
442	44.2	43.9	44.0	45.3
443	38.1	36.3	37.3	38.9
Mean	41.4	40.3	41.7	42.2
STD	2.2	2.9	3.0	2.7
SE	0.8	1.1	1.1	1.0

[a]Chair rest.
[b]Recovery.

Hemoglobin concentration

HDT1			Minutes				
Sub. #	C.R.[a]	1 hr	0	10	20	68	Rec[b]
243	15.9	15.4	15.4	16.9	16.9	16.6	15.8
292	16.1	15.6	15.6	17.6	17.7	17.5	16.6
385	14.4	14.0	14.0	15.5	14.9	15.4	14.8
440	16.6	15.9	15.9	17.4	17.4	17.4	16.2
441	15.8	15.2	15.2	16.6	16.6	17.0	16.4
442	18.0	16.8	16.8	17.2	17.2	19.3	16.9
443	16.5	16.4	16.4	17.6	17.3	17.7	16.7
Mean	16.2	15.6	15.6	17.0	16.9	17.3	16.2
STD	1.0	0.8	0.8	0.7	0.9	1.1	0.7
SE	0.4	0.3	0.3	0.3	0.3	0.4	0.2

HDT24								
Sub. #	C.R.	1 hr	24 hr	0	10	20	68	Rec
243	16.8	16.5	15.9	16.4	17.7	17.5	17.9	17.3
292	17.2	15.6	16.0	16.3	17.6	17.5	17.6	17.0
385	16.2	15.4	15.6	15.6	16.9	16.5	17.0	16.1
440	16.3	16.2	16.3	17.0	19.3	19.6	19.4	18.4
441	15.8	14.7	15.1	17.1	16.9	17.5	17.8	17.1
442	16.3	16.0	17.5	17.6	18.1	18.9	19.4	18.1
443	16.5	15.8	16.1	16.8	17.1	17.6	17.8	17.4
Mean	16.4	15.7	16.1	16.7	17.7	17.9	18.1	17.3
STD	0.4	0.5	0.7	0.6	0.8	1.0	0.8	0.7
SE	0.2	0.2	0.3	0.2	0.3	0.4	0.3	0.3

WI				
Sub. #	C.R.	1 hr	End	Post
243	16.6	15.4	15.8	15.5
292	16.1	15.8	16.0	16.8
385	14.5	13.8	14.5	14.9
440	16.4	15.3	15.0	15.6
441	15.9	15.9	16.6	16.4
442	16.2	15.8	16.6	17.1
443	16.5	14.2	14.8	15.6
Mean	16.0	15.2	15.6	16.0
STD	0.7	0.8	0.8	0.7
SE	0.3	0.3	0.3	0.3

[a]Chair rest.
[b]Recovery.

Rectal temperature

HDT1				Minutes				
Sub. #	0	10	20	30	40	50	60	70
243	36.78	36.90	37.26	37.50	37.62	37.74	37.80	37.86
292	37.02	37.26	37.56	37.74	37.86	37.98	38.04	38.04
385	36.60	36.90	37.38	37.74	37.98	38.10	38.22	38.40
440	36.84	36.90	37.20	37.50	37.62	37.80	37.86	37.92
441	36.60	36.84	37.14	37.38	37.50	37.62	37.62	37.62
442	36.48	36.72	37.08	37.44	37.74	37.86	37.98	38.04
443	36.66	36.96	37.44	37.74	37.92	38.04	38.16	38.22
Mean	36.71	36.93	37.29	37.58	37.75	37.88	37.95	38.01
STD	0.17	0.15	0.16	0.15	0.16	0.16	0.19	0.23
SE	0.06	0.06	0.06	0.06	0.06	0.06	0.07	0.09

HDT24								
Sub. #	0	10	20	30	40	50	60	70
243	36.78	37.02	37.26	37.38	37.56	37.68	37.74	37.80
292	37.50	37.62	37.80	37.98	38.10	38.22	38.28	38.22
385	36.90	37.08	37.50	37.74	38.04	38.28	38.46	38.70
440	37.14	37.38	37.62	37.80	37.98	37.98	38.10	38.22
441	36.96	37.02	37.32	37.62	37.86	38.16	38.34	38.58
442	36.78	37.14	37.50	37.74	37.92	38.04	38.22	38.34
443	36.48	36.66	37.02	37.32	37.56	37.74	37.92	37.98
Mean	36.93	37.13	37.43	37.65	37.86	38.01	38.15	38.26
STD	0.30	0.28	0.24	0.22	0.20	0.21	0.23	0.29
SE	0.11	0.11	0.09	0.08	0.08	0.08	0.09	0.11

Mean skin temperature

HDT1				Minutes					
Sub. #	0	10	20	30	40	50	60	70	Rec[a]
243	30.0	30.4	31.0	31.1	31.3	31.1	31.5	31.6	31.2
292	30.4	30.3	30.2	30.4	30.4	30.6	30.7	30.9	30.6
385	30.4	31.0	30.8	30.9	30.9	31.6	31.5	31.6	30.9
440	30.4	30.8	31.1	31.5	31.8	31.7	31.7	31.7	30.5
441	30.1	30.6	31.5	31.9	31.7	32.0	31.9	32.0	31.3
442	29.9	30.4	30.6	30.6	30.9	30.8	31.2	31.1	30.8
443	29.3	29.7	30.1	31.1	31.3	31.4	31.7	31.8	30.8
Mean	30.1	30.5	30.8	31.1	31.2	31.3	31.5	31.5	30.9
STD	0.4	0.4	0.5	0.5	0.5	0.5	0.4	0.4	0.3
SE	0.1	0.1	0.2	0.2	0.2	0.2	0.1	0.1	0.1

HDT24									
Sub. #	0	10	20	30	40	50	60	70	Rec[a]
243	31.3	31.4	31.5	31.6	31.8	32.0	31.9	31.9	31.9
292	30.6	30.4	30.3	31.0	31.1	31.2	30.7	30.7	30.8
385	30.4	30.9	31.5	31.7	32.1	32.1	32.3	32.2	31.9
440	30.4	31.4	31.5	31.3	31.2	30.5	31.1	30.8	31.2
441	29.5	29.7	31.0	30.9	30.7	30.4	30.3	30.5	31.9
442	30.4	30.7	30.9	30.5	30.5	30.6	29.8	30.5	9.4
443	30.1	30.1	30.7	31.1	31.1	31.2	31.3	31.2	31.2
Mean	30.4	30.7	31.1	31.2	31.2	31.1	31.1	31.1	31.2
STD	0.5	0.6	0.4	0.4	0.5	0.6	0.8	0.6	0.8
SE	0.2	0.2	0.2	0.1	0.2	0.2	0.3	0.2	0.3

[a]Recovery.

LaserFlo Doppler velocity (Hz × 10^2)

HDT1				Minutes				
Sub. #	0	10	20	30	40	50	60	70
243	0.49	0.62	1.56	0.83	1.70	0.34	1.38	1.21
292	0.44	0.92	1.03	1.03	1.04	1.05	1.08	0.78
385	0.53	1.16	1.36	2.03	2.35	2.45	2.18	2.06
440	0.45	1.02	1.96	2.25	2.47	2.26	2.14	2.09
441	0.33	0.96	1.37	1.26	1.54	1.70	2.04	2.14
442	0.41	0.95	0.96	0.96	0.93	1.02	1.01	1.07
443	0.38	0.49	0.92	1.96	2.04	1.45	1.63	1.57
Mean	0.43	0.87	1.31	1.47	1.72	1.47	1.64	1.56
STD	0.06	0.22	0.35	0.54	0.56	0.69	0.46	0.51
SE	0.02	0.08	0.13	0.21	0.21	0.26	0.17	0.19

HDT24								
Sub. #	0	10	20	30	40	50	60	70
243	0.66	1.13	1.99	2.16	2.02	1.97	1.76	1.65
292	0.52	0.67	0.88	0.80	0.95	1.09	1.22	1.10
385	0.59	1.47	4.03	5.10	4.40	4.48	3.84	3.08
440	1.56	1.78	1.88	1.44	1.64	1.50	1.35	1.54
441	0.35	1.60	1.66	1.84	1.86	1.82	1.86	2.12
442	0.36	0.55	2.28	2.56	2.14	1.86	1.31	1.08
443	0.58	0.71	1.18	1.22	1.17	1.10	0.96	0.99
Mean	0.66	1.13	1.99	2.16	2.02	1.97	1.76	1.65
STD	0.41	0.50	1.02	1.42	1.13	1.16	0.97	0.75
SE	0.17	0.20	0.42	0.58	0.46	0.47	0.40	0.31

Exercise heart rates

HDT1			Minutes				
Sub. #	10	20	30	40	50	60	70
243	109	121	121	129	131	131	131
292	138	138	145	144	151	151	150
385	135	144	145	147	151	155	160
440	122	131	138	137	143	146	146
441	124	126	128	128	132	128	130
442	112	126	130	135	140	147	152
443	124	138	138	140	149	148	155
Mean	123	132	135	137	142	144	146
STD	10	8	8	7	8	9	11
SE	4	3	3	3	3	4	4

HDT24							
Sub. #	10	20	30	40	50	60	70
243	111	114	120	127	130	131	131
292	119	130	135	136	142	147	152
385	139	144	154	159	161	164	173
440	140	146	151	154	153	161	156
441	123	128	140	152	161	165	172
442	124	138	144	148	151	153	154
443	123	130	139	134	137	142	148
Mean	126	133	140	144	148	152	155
STD	10	10	10	11	11	12	13
SE	4	4	4	4	4	4	5

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12a. DISTRIBUTION/AVAILABILITY STATEMENT

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13. ABSTRACT ***(Maximum 200 words)***

The hypothesized fluid shifts and resultant responses that occur during spaceflight are simulated by six-degree head-down tilt (HDT) and water immersion (WI). The purpose of this study was to compare exercise thermoregulation before and after physiologic mechanisms reduce plasma volume (PV) in response to 24-hour (hr) HDT (HDT24). A secondary study utilized WI to reproduce the PV reduction of HDT24. Seven males were studied in two conditions: 1) during 70 minutes of supine cycling ergometry at 58% of peak oxygen consumption following 1-hr HDT (HDT1) and HDT24; and 2) up to 6 hr WI at 34.5 degrees Centigrade (°C).

Plasma volume was reduced by 10.4% in HDT24 when compared to HDT1. Preexercise rectal temperature (T_{re}) was an average 0.22°C higher after HDT24. Rectal temperature increased during exercise with no interaction between time and treatment. The reduced PV and elevated preexercise T_{re} had offsetting effects on thermoregulatory mechanisms, suggesting no alteration in the response at a given T_{re}.

Plasma volume was reduced by 4.3 ± 2.3% and 1.1 ± 1.8% following HDT24 and WI, respectively, compared to upright chair rest. Although the reductions in PV were not significantly different, great intraindividual variability was evident. The ability to reproduce PV changes consistently with HDT and WI is limited by this variability.

14. SUBJECT TERMS

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